An Evaluation of Transcutaneous Carbon Dioxide Partial Pressure Monitoring during Apnea Testing in Brain-dead Patients

Benoît Vivien, M.D., Ph.D.,* Frédéric Marmion, M.D.,* Sabine Roche, M.D.,* Catherine Devilliers, Sc.D.,† Olivier Langeron, M.D., Ph.D.,* Pierre Coriat, M.D.,‡ Bruno Riou, M.D., Ph.D.§

Background: Diagnosis of brain death usually requires an arterial carbon dioxide partial pressure (Paco2) of 60 mmHg during the apnea test, but the increase in Paco2 is unpredictable. The authors evaluated whether transcutaneous carbon dioxide partial pressure (PtcCO2) monitoring during apnea test can predict that a Paco2 of 60 mmHg has been reached.

Methods: The authors compared PtcCO2 measured with a transcutaneous ear sensor (V-Sign® Sensor, Sentec Digital Monitoring System; SENTEC-AG, Therwil, Switzerland) and Paco2 obtained from arterial blood gas measurements in 32 clinically brain-dead patients.

Results: In the first 20 patients, the mean Paco2-PtcCO2 gradient was 0.7 ± 3.6 mmHg at baseline and 8.7 ± 7.1 mmHg after 20 min of apnea. Using receiver operating characteristic curve analysis (area under the curve: 0.983 ± 0.013), the best threshold value of PtcCO2 to predict that a Paco2 of 60 mmHg had been reached was 60 mmHg (positive predictive value: 1.00 [0.93–1.00]). In the following 12 patients investigated with use of this PtcCO2 target value of 60 mmHg, the mean duration of the apnea test (11 ± 4 vs. 20 ± 0 min; P < 0.001), hypcapnia (74.0 ± 4 vs. 98.3 ± 20.0 mmHg; P < 0.001), acidosis (pH: 7.18 ± 0.06 vs. 7.11 ± 0.08; P < 0.001), and decrease in arterial oxygen partial pressure (−47 ± 44 vs. −95 ± 89; P < 0.05) at the end of the test were reduced as compared with the 20-min apnea test group.

Conclusion: During the apnea test in brain-dead patients, a PtcCO2 of 60 mmHg accurately predicts that a Paco2 of 60 mmHg has been reached. This may allow a reduction in the duration of the apnea test and consecutively limit occurrence of complications.

BRAIN death is defined by the irreversible cessation of all cortical functions, including the brainstem reflexes, motor responses, and respiratory drive, in a normothermic, un sedated, comatose patient with an irreversible major brain injury and a noncontributing metabolic disorder.1 When brain death is clinically suspected, an important component of the clinical diagnosis is the apnea test, although this is not always required by guidelines and/or law throughout the world.2 An arterial carbon dioxide partial pressure (Paco2) target value of 60 mmHg at the end of the apnea test is usually recommended.3 However, during the apnea test in brain-dead patients, the estimated Paco2 increase is slow, from 1.7 to 3.7 ± 2.3 mmHg/min, and biphasic with a decline in the increase rate throughout the duration of the apnea test.4–9 In addition, the increase in Paco2 has been reported as unpredictable, from 0.5 to 10.5 mmHg/min, because of carbon dioxide washout, atelectasis, cardiac-induced ventilations, and other potentially unknown factors,6 which explains the failure of attempts to estimate the required duration of the apnea test to reach the threshold of a Paco2 of 60 mmHg.4,10

Transcutaneous carbon dioxide partial pressure monitoring (PtcCO2), which has been used for several decades in infants, is now a valid technique in adults and provides noninvasive, accurate, and real-time monitoring of Paco2 and allows a significant reduction in, but does not replace, arterial samples for blood gas analysis.11–17 However, although PtcCO2 monitoring has been reported to be in good agreement with Paco2 during stable ventilatory and circulatory conditions both in volunteers and in anesthetized patients, it had been reported that the accuracy of this monitoring became more imprecise during major increases in Paco2, such as the apnea test in brain-dead patients.17 Indeed, Lang et al. had previously studied PtcCO2 monitoring during an apnea test in brain-dead patients, but because they induced an increase in Paco2 either by hyperventilation or by artificial carbon dioxide augmentation, both followed by a real apnea time of only 0.5–1 min, they overlooked the dynamic component of the Paco2 increase during the apnea.17,18 Moreover, this apnea test procedure performed in their two studies was not the one commonly recommended throughout the world, which usually requires a starting arterial Paco2 of 40 mmHg before disconnection from the ventilator.1,3

Therefore, the aim of this prospective clinical study was first to evaluate the accuracy of PtcCO2 monitoring as a real-time estimate of Paco2 during the apnea test in brain-dead patients and second to determine whether PtcCO2 monitoring could accurately predict that the Paco2 target value of 60 mmHg has been reached, therefore enabling shortening of the duration of the apnea test.

Materials and Methods

Study Population

The study was approved by our local ethics committee (Comité de Protection des Personnes se Prêtant à la...
Recherche Biomédicale, Groupe Hospitalier Pitié-Salpêtrière, Paris, France). Thirty-two patients clinically suspected of being brain dead were investigated prospectively during a 7-month period (December 2004 to June 2005). All of them had been admitted to the intensive care unit (ICU) for severe coma resulting mainly from spontaneous intracranial hemorrhage, head injury, or cerebral anoxia. The cause of coma was established for every patient, and reversible abnormalities (drug and metabolic intoxications, hypothermia < 35°C, and shock) were excluded. Because of the severity of their cerebral lesions at the time of admission into the ICU, all of these patients were potentially expected to develop brain death. Care of the patients conformed to standard procedures in our ICU for severely comatose patients. The patients were monitored with an arterial pressure catheter, enabling samples to be taken for arterial blood gas measurements. PtcCO₂ was continuously measured with a heated transcutaneous ear sensor (V-Sign® Sensor, Sentec Digital Monitoring System; SENTEC-AG, Therwil, Switzerland), which also combines a pulse oximetry sensor. The PtcCO₂ measurement by the V-Sign® Sensor is based on a Severinghaus-type electrochemical carbon dioxide tension sensor. The sensor temperature is warmed up to 42°C to achieve local arterialization of the skin at the monitoring site for the PtcCO₂ measurement. According to the manufacturer’s recommendations, the sensor was automatically calibrated in vitro in its integrated calibration unit (“docking station”) before each apnea test. After calibration, the sensor was applied to the patient’s ear lobe using a single-use ear clip and a thin layer of sensor gel, and a 20-min equilibration time was allowed before measurements.

At the time of investigation, all patients were in a deep, unresponsive coma. They lacked all bulbar reflexes (pupillary [light], corneal, oculocardiac, and oropharyngeal [gag and cough] reflexes), had no spontaneous breathing movements, and usually showed vasoplegia and diabetes insipidus. All these findings strongly indicate brain death.19 Because the apnea test has been shown to be deleterious in some patients and may therefore limit organ procurement for transplantation,9,20–22 we have decided in our ICU to perform the apnea test only after brain death has been confirmed by electrocortical silence on one electroencephalogram with maximal amplification. On the other hand, when the electroencephalogram is unhelpful in confirming brain death (mainly because of hypothermia < 35°C or because of a significant residual blood concentration of sedative drugs), we usually require the absence of intracerebral blood flow on four-vessel cerebral angiography. However, angiography is not only potentially deleterious, but also risky because of transportation of the patient to the radiology department, especially when there is major hemodynamic instability.23,24 Therefore, when four-vessel cerebral angiography is mandatory, we usually perform the apnea test before angiography. In such cases, before the apnea test, we always verify the absence of intracerebral blood flow by transcranial Doppler ultrasonography.3

The Apnea Test

The apnea test was performed after a 20-min preoxygenation period with an inspired oxygen fraction of 100%. After the ventilator was disconnected, a 94/min oxygen flow was delivered through the endotracheal tube via an oxygen cannula (12-French catheter). The patient was then closely observed for respiratory efforts. If spontaneously respiratory efforts or complications (major hemodynamic instability despite increase in the dose of catecholamine and/or severe hypoxemia) occurred, the apnea was discontinued and the patient was immediately reconnected to the ventilator. Otherwise, the apnea was continued, and afterward, the patient was reconnected to the ventilator at the end of the test. The apnea test was considered positive if there was no respiratory effort and if PaCO₂ reached at the end of the test was 60 mmHg or higher.3

In the first 20 brain-dead patients (20-min apnea test group), the apnea test was performed according to our standard guideline, i.e., over a 20-min fixed period. Using the receiver operating characteristic (ROC) curve, this enabled us to calculate the best PtcCO₂ target value which estimates that the PaCO₂ threshold of 60 mmHg has been reached. Thereafter, for the following 12 brain-dead patients (PtcCO₂ targeted apnea test group), the apnea test was performed until this previously calculated PtcCO₂ target had been reached.

Data Collection

Clinical characteristics, etiology of brain death, and hemodynamic variables (heart rate, systolic arterial blood pressure, and oxygen peripheral saturation) were recorded. PtcCO₂ was continuously monitored before, during, and after the apnea test, and data were stored on a computer for off-line analysis. Complications during the apnea test were recorded as hypotension (defined as a decrease in systolic arterial blood pressure of more than 20% of baseline value and/or the need for an increase in the dose of catecholamine administered), hypertension (defined as an increase in arterial blood pressure of more than 20% of baseline value and/or the need for a decrease in the dose of catecholamine administered), and severe hypoxemia (defined as a decrease of oxygen peripheral saturation < 90%). For PaCO₂ measurements (Blood Gas Analyzer ABL725; Radiometer, Copenhagen, Denmark), arterial blood gases were obtained through an indwelling radial arterial line and were compared with the simultaneous PtcCO₂. In the 20-min apnea test group, arterial blood gases were sampled before the apnea test (baseline) and thereafter at 5, 10, 15, and 20 min of apnea. In the PtcCO₂ targeted apnea test group, arterial blood gases were sampled before the
apnea test (baseline) and thereafter at the end of the apnea test when the Ptc CO2 had reached the calculated target value. In addition, after reconnection of the patient to the ventilator at the end of the apnea test, arterial blood gases were sampled at 5-min intervals for 30 min in 10 patients of the 20-min apnea test group.

**Statistical Analysis**

Data are expressed as mean ± SD. Comparison of two means was performed using the Student t test. The ROC curve was used to determine the best threshold value for PtCO2 to predict that PaCO2 has reached the mandatory threshold of 60 mmHg. The area under the ROC curve was also calculated. Sensitivity, specificity, positive and negative predictive values, accuracy (defined as the sum of concordant cells divided by the sum of all cells in the two-by-two table), and their 95% confidence intervals were calculated. The best threshold value was defined as the one that simultaneously minimizes the distance to the ideal point (sensitivity = specificity = 1) and that provides a positive predictive value as close as possible to 1. All P values were two tailed, and a P value of less than 0.05 was considered significant. The NCSS 2001 statistical program (Statistical Solutions Ltd., Cork, Ireland) was used for all statistical analyses.

**Results**

The apnea test was performed 32 times in 32 patients, 24 men and 8 women (mean age, 48 ± 14 yr). Causes of brain death were cerebral hemorrhage (n = 20), blunt head trauma (n = 5), cerebral anoxia (n = 4), and cerebral gunshot injury (n = 3). At the time of investigation, 31 patients required catecholamine administration, and 23 patients exhibited diabetes insipidus. The confirmatory test of brain death was electroencephalogram in 19 patients, whereas the 13 other patients required cerebral angiography because of significant residual blood concentration of sedative drugs. The apnea test was completely performed in the 20 patients of the 20-min apnea test group, and none of them showed any spontaneous respiratory movement during the 20-min apnea period. Similarly, in the 12 patients of the PtcCO2 targeted apnea test group, the apnea test was performed until PtcCO2 had reached the calculated target value, and none of them showed any spontaneous respiratory movement during apnea. Fourteen patients in the 20-min apnea test group and 4 patients in the PtcCO2 targeted apnea test group showed a significant hypotension requiring an increase in the dose of catecholamine administered (P < 0.05). Finally, whatever the apnea test group, none of the 32 investigated patients showed significant hypoxemia during the apnea test.

The mean PaCO2–PtcCO2 gradient was 0.7 ± 3.6 mmHg for baseline measurement before the apnea test in the 32 investigated patients. Figure 1 presents the typical recording of PaCO2 and PtcCO2 during the apnea test in one patient of the 20-min apnea test group. Arterial carbon dioxide partial pressure (PaCO2) values are plotted for comparison to the simultaneous PtcCO2. PtcCO2 was very close to PaCO2 for baseline measurement but exhibited a certain “delay” during the rapid increase of PaCO2 during apnea. After reconnection to the ventilator, PtcCO2 became closer to PaCO2 as the rate of decrease in PaCO2 was reduced. This hemodynamically stable brain-dead patient did not present any complication during the 20 min of the apnea test.

The mean PaCO2–PtcCO2 gradient was 0.7 ± 3.6 mmHg for baseline measurement before the apnea test in the 32 investigated patients. Figure 1 presents the typical recording of PaCO2 and PtcCO2 during the apnea test in one patient of the 20-min apnea test group. The increases in PaCO2 and PtcCO2 during the apnea test in the 20-min apnea test group are shown on figure 2. During the apnea test, the mean PaCO2–PtcCO2 gradient was fairly stable, approximately 8.7 ± 7.1. The box plot representation of the PaCO2–PtcCO2 gradient clearly shows that the gradient observed during the apnea test could not be analyzed in the same manner as the baseline gradient.

The mean PaCO2–PtcCO2 gradient was 0.7 ± 3.6 mmHg for baseline measurement before the apnea test in the 32 investigated patients. Figure 1 presents the typical recording of PaCO2 and PtcCO2 during the apnea test in one patient of the 20-min apnea test group. Arterial carbon dioxide partial pressure (PaCO2) values are plotted for comparison to the simultaneous PtcCO2. PtcCO2 was very close to PaCO2 for baseline measurement but exhibited a certain “delay” during the rapid increase of PaCO2 during apnea. After reconnection to the ventilator, PtcCO2 became closer to PaCO2 as the rate of decrease in PaCO2 was reduced. This hemodynamically stable brain-dead patient did not present any complication during the 20 min of the apnea test.

The mean PaCO2–PtcCO2 gradient was 0.7 ± 3.6 mmHg for baseline measurement before the apnea test in the 32 investigated patients. Figure 1 presents the typical recording of PaCO2 and PtcCO2 during the apnea test in one patient of the 20-min apnea test group. Arterial carbon dioxide partial pressure (PaCO2) values are plotted for comparison to the simultaneous PtcCO2. PtcCO2 was very close to PaCO2 for baseline measurement but exhibited a certain “delay” during the rapid increase of PaCO2 during apnea. After reconnection to the ventilator, PtcCO2 became closer to PaCO2 as the rate of decrease in PaCO2 was reduced. This hemodynamically stable brain-dead patient did not present any complication during the 20 min of the apnea test.
The Bland-Altman analysis for comparison of PtcCO₂ versus PaCO₂ during the 20-min apnea test (i.e., excluding baseline measurements) revealed a mean bias of 8.6 mmHg, with limits of agreement (± 1.96 · SD) of 22.4 and −5.3 mmHg (fig. 3B).²⁵ There was no significant correlation between the PaCO₂–PtcCO₂ gradient and the rate of increase in PaCO₂ during the apnea (R = 0.19). At the end of the 20-min apnea test, we observed major hypercapnia, acidosis, and decrease in PaO₂ as compared with baseline measurement (table 1). During the first 30 min after reconnection of the patient to the ventilator, the mean PaCO₂–PtcCO₂ gradient progressively increased from 1.8 ± 11.5 mmHg to 8.5 ± 6.0 mmHg (fig. 2). The capacity of PtcCO₂ to predict that the targeted PaCO₂ 60 mmHg had been reached was assessed with a ROC curve analysis (fig. 4). The area under the ROC curve was 0.983 ± 0.013, indicating a very high accuracy of PtcCO₂ monitoring. The best threshold value on receiver operating characteristic curve analysis is the one that simultaneously minimizes the distance to the ideal point (sensitivity = specificity = 1) and that provides a positive predictive value as close as possible to 1.

Table 1. Arterial Blood Gases before and at the End of the Apnea Test for the 20-min Apnea Test Group and the PtcCO₂ Targeted Apnea Test Group

<table>
<thead>
<tr>
<th></th>
<th>20-min Apnea Test (n = 20)</th>
<th>PtcCO₂ Targeted Apnea Test (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>End of Apnea</td>
</tr>
<tr>
<td>Pao₂, mmHg</td>
<td>353 ± 98</td>
<td>268 ± 121*</td>
</tr>
<tr>
<td>ΔPao₂, mmHg</td>
<td>—</td>
<td>−95 ± 89</td>
</tr>
<tr>
<td>SaO₂, %</td>
<td>99.7 ± 0.8</td>
<td>96.9 ± 10.8*</td>
</tr>
<tr>
<td>pH</td>
<td>7.40 ± 0.08</td>
<td>7.11 ± 0.08*</td>
</tr>
<tr>
<td>HCO₃, mm</td>
<td>25.0 ± 2.7</td>
<td>28.9 ± 3.8*</td>
</tr>
<tr>
<td>Paco₂, mmHg</td>
<td>41.4 ± 6.3</td>
<td>98.3 ± 20.0*</td>
</tr>
<tr>
<td>PtcCO₂, mmHg</td>
<td>41.1 ± 5.8</td>
<td>88.6 ± 20.0*</td>
</tr>
<tr>
<td>Paco₂ – PtcCO₂, mmHg</td>
<td>0.3 ± 4.2</td>
<td>9.7 ± 9.0*</td>
</tr>
</tbody>
</table>

Data are mean ± SD.

* P < 0.05 vs. baseline in the same apnea test group. † P < 0.05 vs. end of apnea in the 20-min apnea test group.

Paco₂ = arterial carbon dioxide partial pressure; Pao₂ = arterial oxygen partial pressure; ΔPao₂ = arterial oxygen partial pressure in baseline conditions minus arterial oxygen partial pressure at the end of the apnea test; PtcCO₂ = transcutaneous carbon dioxide partial pressure; SaO₂ = oxygen saturation.
1.00 [0.93–1.00], a negative predictive value of 0.72 [0.57–0.83], and an accuracy of 0.87 [0.78–0.92] (table 2).

Therefore, the 12 patients of the PtcCO2 targeted apnea test group were investigated using this calculated PtcCO2 target value of 60 mmHg. The mean duration of the PtcCO2 targeted apnea test was significantly reduced: 11±4 min (P < 0.001 vs. 20 ± 0 min). None of the 12 patients exhibited a PaCO2 lower than 60 mmHg at the end of the apnea test (extreme values: 64.1–79.4 mmHg).

The mean PaCO2–PtcCO2 gradient at the end of the apnea test in the PtcCO2 targeted apnea test group was 12.0 ± 4.6 mmHg (table 1). Figure 5 presents the typical recording of PaCO2 and PtcCO2 during the apnea test in one patient of the PtcCO2 targeted apnea test group. As expected, table 1 shows that hypercapnia, acidosis, and decrease in PaO2 at the end of the apnea test were significantly reduced in the PtcCO2 targeted apnea test as compared with the 20-min apnea test group.

**Discussion**

In this study, we have shown that PtcCO2 monitoring during the apnea test in brain-dead patients (1) exhibits a mean PaCO2–PtcCO2 gradient of 8.7 ± 7.1 mmHg that is fairly stable during a 20-min apnea test; and (2) could accurately predict that the target PaCO2 of 60 mmHg had been reached, therefore enabling us to shorten the apnea test and thus significantly reduce hypercapnia, acidosis, and decrease in PaO2 at the end of the apnea test.

The mean PaCO2–PtcCO2 gradient was 0.7 ± 3.6 mmHg for the baseline measurement before the apnea test in the 32 brain-dead patients. This close agreement between PaCO2 and PtcCO2 had been previously reported both in volunteers and in anesthetized patients.11–16 However, most of these studies investigated PtcCO2 monitoring at equilibrium during stable ventilatory and circulatory conditions. Conversely, during the apnea test, we observed that the mean PaCO2–PtcCO2 gradient was fairly stable, approximately 8.7 ± 7.1 mmHg in the 20-min apnea test group, and 12.0 ± 4.6 mmHg in the PtcCO2 targeted apnea test group. This increased gradient in such dynamic conditions is fully understandable because of the continuous increase in PaCO2 during the apnea. Indeed, the “delay” of PtcCO2 monitoring as compared with the simultaneous PaCO2 measured by blood gas analysis could be linked to the time for diffusion of carbon dioxide from the ear lobe capillary to the sensor, and eventually the time of calculation of PtcCO2 by the monitor. At last, the slightly higher mean PaCO2–PtcCO2 gradient in the PtcCO2 targeted apnea test group as compared with the 20-min apnea test group could probably be explained by the decline in the increase rate in PaCO2 throughout the duration of the apnea test.6–8

The mean rate of increase in PaCO2 during the 20-min apnea test was 2.9 ± 0.8 mmHg/min in our study, close to those reported by Ropper et al.4 (2.6 ± 0.8 mmHg/min) and Orliaguet et al.7 (2.7 mmHg/min). We did not find a significant correlation between the PaCO2–PtcCO2 gradient and the rate of increase in PaCO2 during the apnea. On one hand, the PaCO2 increase is highly variable among brain-dead patients during the apnea test because of carbon dioxide washout, atelectasis, cardiac-induced
ventilations, and other potentially unknown factors. On the other hand, the instantaneous $\text{Paco}_2$–$\text{PtcCO}_2$ gradient during such dynamic conditions probably depends on many factors, such as the rate of increase in $\text{Paco}_2$, but also hemodynamic, temperature, ear lobe vascularization, and skin condition. At last, 5 min after reconnection of the patient to the ventilator, $\text{PtcCO}_2$ was very close to $\text{Paco}_2$, likely because the rapid decrease in $\text{Paco}_2$ overtook the delay of $\text{PtcCO}_2$ variations (fig. 2). Afterward, the late increase in the mean $\text{Paco}_2$–$\text{PtcCO}_2$ gradient is unexpected and could be due to a drift of the transcutaneous sensor because of the extreme and rapid changes in $\text{Paco}_2$ during the apnea test and reconnection to the ventilator.

The area under the ROC curve was 0.983 ± 0.013, indicating a very high accuracy of $\text{PtcCO}_2$ in predicting that the target $\text{Paco}_2$ 60 mmHg had been reached (fig. 4). The best threshold value on ROC curve analysis was determined as a $\text{PtcCO}_2$ of 60 mmHg, providing a sensitivity of 0.80, a specificity of 1.00, and a positive predictive value of 1.00 (table 2). Indeed, none of the 12 patients of the $\text{PtcCO}_2$ targeted apnea test group investigated using this threshold of 60 mmHg exhibited a $\text{Paco}_2$ lower than 60 mmHg at the end of the apnea test. In a previous study investigating $\text{PtcCO}_2$ monitoring for apnea testing in brain-dead patients, Lang17 showed that a $\text{PtcCO}_2$ of 66 mmHg had a predictive value of 82% for $\text{Paco}_2$ of 60 mmHg or greater and empirically recommended a $\text{PtcCO}_2$ of 60–66 mmHg for the confirmatory arterial blood gas check. This discrepancy as compared with our result may be fully explained, because in their analysis of the $\text{Paco}_2$–$\text{PtcCO}_2$ gradient, they overlooked the dynamic component of $\text{Paco}_2$ increase during the apnea. Finally, the apnea test procedures performed in their study were either hypoventilation or artificial carbon dioxide insufflation, with a real apnea time of only 0.5–1 min, i.e., far from the apnea test procedure commonly recommended throughout the world.1,3

For the 12 patients of the $\text{PtcCO}_2$ targeted apnea test group, the mean duration of the apnea test, hypercapnia, acidosis, and decrease in $\text{Pao}_2$ at the end of the apnea test were significantly reduced as compared with the 20-min apnea test group. This result is important, because performing an apnea test in brain-dead patients may lead to complications, such as hypotension and hypoxia, especially in patients with hemodynamic instability. In the worst possible situation, the apnea test may exceptionally induce a sudden and irreversible cardiac arrest, which prevents any organ donation.9,27,28 Limitation of the duration of the apnea test reduces the importance of hypercapnia, acidosis, and decrease in $\text{Pao}_2$ at the end of the apnea and therefore probably reduces the occurrence of complications related to the test. Indeed, we observed significantly less hypotension in the $\text{PtcCO}_2$ targeted apnea test group than in the 20-min apnea test group. On the other hand, whatever the apnea test group, none of our 32 patients exhibited severe hypoxemia during the apnea test. Indeed, the 20-min preoxygenation period with an inspired oxygen fraction of 100% and the 9 l/min oxygen insufflation inside the endotracheal tube during the apnea test, both performed in our study, probably limited the occurrence of significant hypoxemia during the apnea test.1,9,29 Nevertheless, further studies are mandatory to determine whether reduction of the duration of the apnea test in brain-dead patients may lead to a significant improvement in the prognosis of transplanted organs.

Finally, one could argue that the 20-min apnea test period we have chosen for the first 20 investigated patients was dramatically too long, because the mean $\text{Paco}_2$ at 5 min of the apnea test (63.8 ± 10.1 mmHg, fig. 2) in the 20-min apnea test group was already higher than the threshold of 60 mmHg and because the mean duration of the apnea test in the $\text{PtcCO}_2$ targeted apnea test group was reduced to 11 ± 4 min. However, the $\text{Paco}_2$ increase during the apnea test in brain-dead patients is unpredictable from one patient to another, and shorter apnea test times, such as 10 min, have been previously reported as insufficient in some patients to reach the $\text{Paco}_2$ threshold value of 60 mmHg.6,9,30 Similarly, estimation of the required apnea test duration to reach the threshold of 60 mmHg has also been reported as inefficient because of the unpredictability of $\text{Paco}_2$ increase during the apnea test.4,10 This explains why some investigators, who had reported an apnea test lasting from 1 min to more than 1 h, strongly discourage a time-locked approach for the apnea test and conversely insist on arterial blood gas determinations.31 On the other hand, keeping in mind the dynamic $\text{Paco}_2$–$\text{PtcCO}_2$ gradient during the apnea test, $\text{PtcCO}_2$ monitoring offers an on-line estimation of $\text{Paco}_2$, whereas $\text{Paco}_2$ analysis from a blood gas sample requires a minimum delay of several minutes to get the result, and eventually a longer time depending on the distance between the laboratory and the ICU.32 Nevertheless, one should keep in mind that the high predictive value of $\text{PtcCO}_2$ reported in our study with the V-Sign® Sensor may not be found with other transcutaneous carbon dioxide sensors, according to technology and time–response differences between devices.

In conclusion, $\text{PtcCO}_2$ monitoring during the apnea test in brain-dead patients may permit a significant reduction in the duration of the apnea test. We found that a $\text{PtcCO}_2$ of 60 mmHg has a predictive positive value of 100% in predicting that the target threshold of $\text{Paco}_2$ of 60 mmHg has been reached. Reducing the duration of the apnea test may limit hypercapnia, acidosis, and decrease in $\text{Pao}_2$ at the end of the apnea test and eventually occurrence of complications such as hypoxemia and hypotension.
References


Anesthesiology, V 104, No 4, Apr 2006